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CURRENT PROGRESS

Neurophthalmological Aspects of Tumours of the Third Ventricle

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THE symptomatology which may be associated with tumours arising within or invading the third ventricle is extremely variable. Such symptomatology is vitally important for neurological and neurophthalmological diagnoses. New information will not be found in this study. Although more than 200 cases have been reviewed, no effort has been made to cover all the literature on the subject. Sixteen personal cases are included because of interesting histories or pathological findings which exemplify the presentation. A brief summary of the basic embryology, anatomy and physiology involved is included to permit a more complete discussion of clinical symptomatology.

I. The Embryological and Anatomical Basis of Clinical Symptomatology

Embryological Origin of Tumours

Three embryologically different tissues may be involved in the appearance of tumours in the region of the third ventricle. Besides neurectoderm and mesoderm we find ectoderm derived from Rathke's pouch during the formation of the anterior lobe of the hypophysis. Remnants of this ectoderm are believed to be related to the appearance of craniopharyngiomas. The paraphysis, a gland found in certain animals, regresses entirely in man. It would be situated at the level of the foramina of Monro. Remnants of this gland, seen as choroidal folds, are believed to be responsible for the colloid cysts found in this region.[1-3]

These facts suggest that a great many types of tumours can be expected in this region. A brief summary is given in Tables I and II. We have included for the sake of interest a few references[4-17] and personal cases (Figs. 1-6).

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TABLE I.—TUMOURS OF THE THIRD VENTRICLE ARISING FROM VENTRICULAR STRUCTURES AND THEIR ORIGIN

Walls of the ventricle:	*Type of tumour:*
Ependymal layer	Ependymomas
Supporting tissues	Gliomas
	Tuberous sclerosis
	Intraventricular epidermoids
Ventricular structures:	
Choroid plexus	Papillomas
Paraphysis, choroidal folds	Colloid cysts

Anatomical Basis for Changes Produced by Tumours

Tumours of the third ventricle give rise to symptoms through compression or invasion of adjacent structures. It is convenient to subdivide these into five regions surrounding the ventricle (Fig. 7).

Region 1: Anteroinferior structures.—Bounded laterally by the hypothalamus, the anteroinferior part of the ventricle ends in a cul-de-sac, actually in direct contact with the optic chiasm, the optic recess. We must recall the presence of the third nerves originating in the interpeduncular space and running close to this region.

Region 2: Anterosuperior structures.—The only capital structures here are the foramina of Monro.

Region 3: Inferior structures.—The floor of the ventricle contains many structures: the third

TABLE II.—TUMOURS AND NON-TUMORAL LESIONS ARISING FROM EXTRAVENTRICULAR STRUCTURES

Tumours growing into the third ventricle
- Extrasellar pituitary adenomas
- Epidermoid cysts and tumours
- Craniopharyngiomas
- Infundibulomas
- Pineal gland—pinealomas, teratomas
- Hamartomas
- Meningiomas
- Lipomas

Non-tumoral lesions that can compress the ventricle
- Midline hematomas
- Dilated basilar arteries
- Tuberculous granulomas

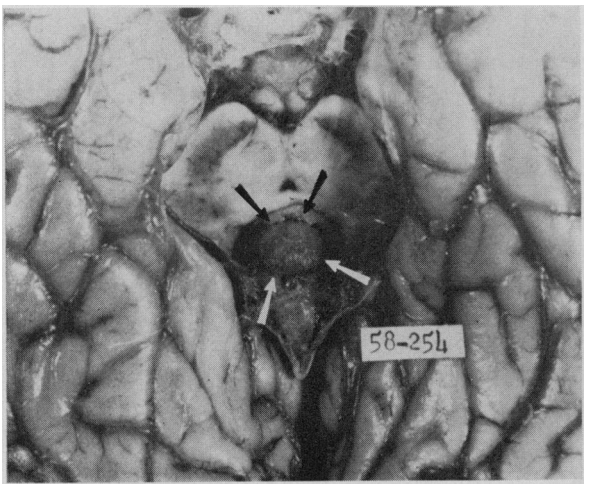


Fig. 1.—Pinealoma.

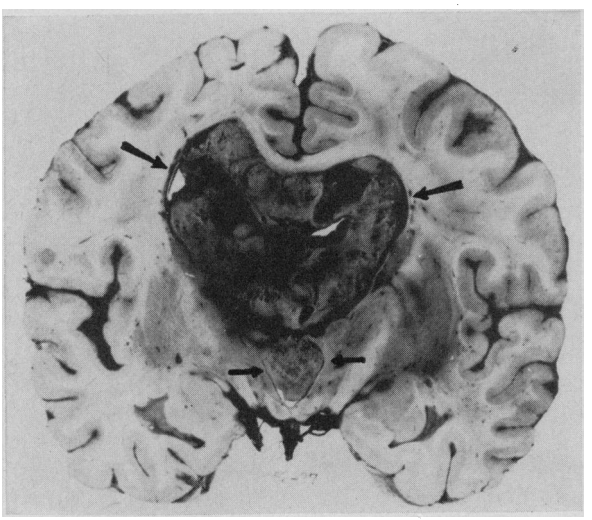


Fig. 2.—Sub-ependymal astrocytoma.

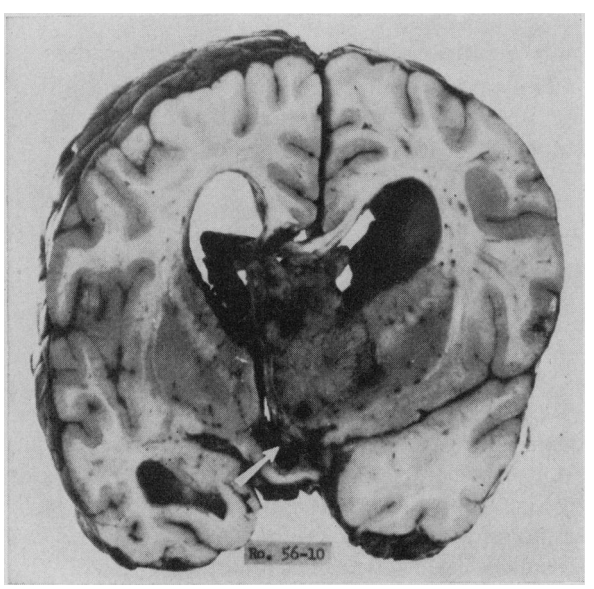


Fig. 3.—Tuberous sclerosis.

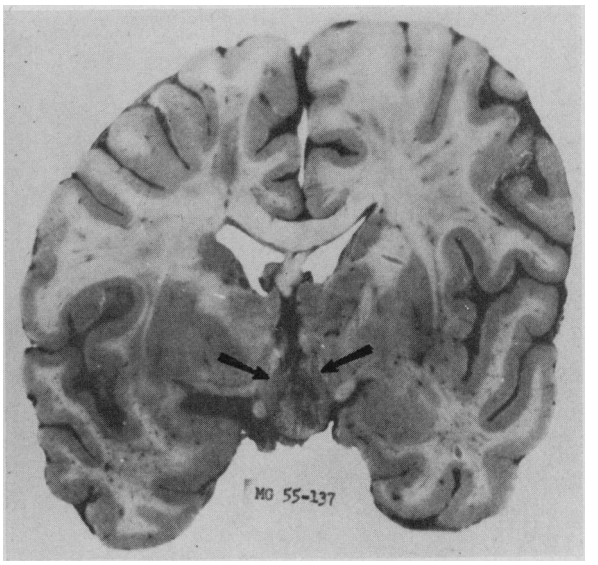


Fig. 4.—Bronchogenic metastasis to tuber cinereum.

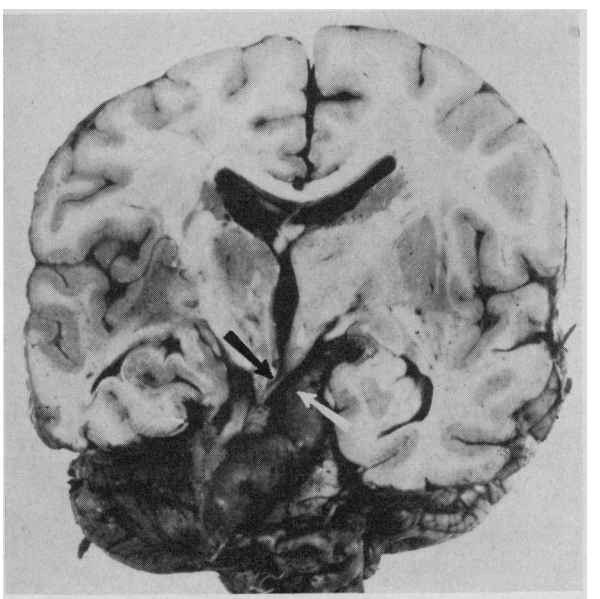


Fig. 5

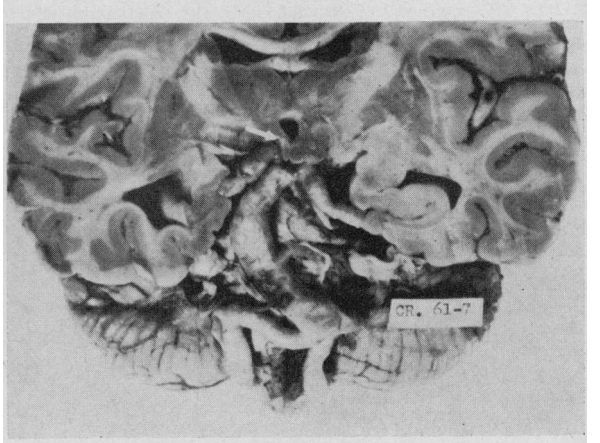


Fig. 6

Figs. 5 and 6.—Two cases of dilated basilar arteries causing distortion of the third ventricle.

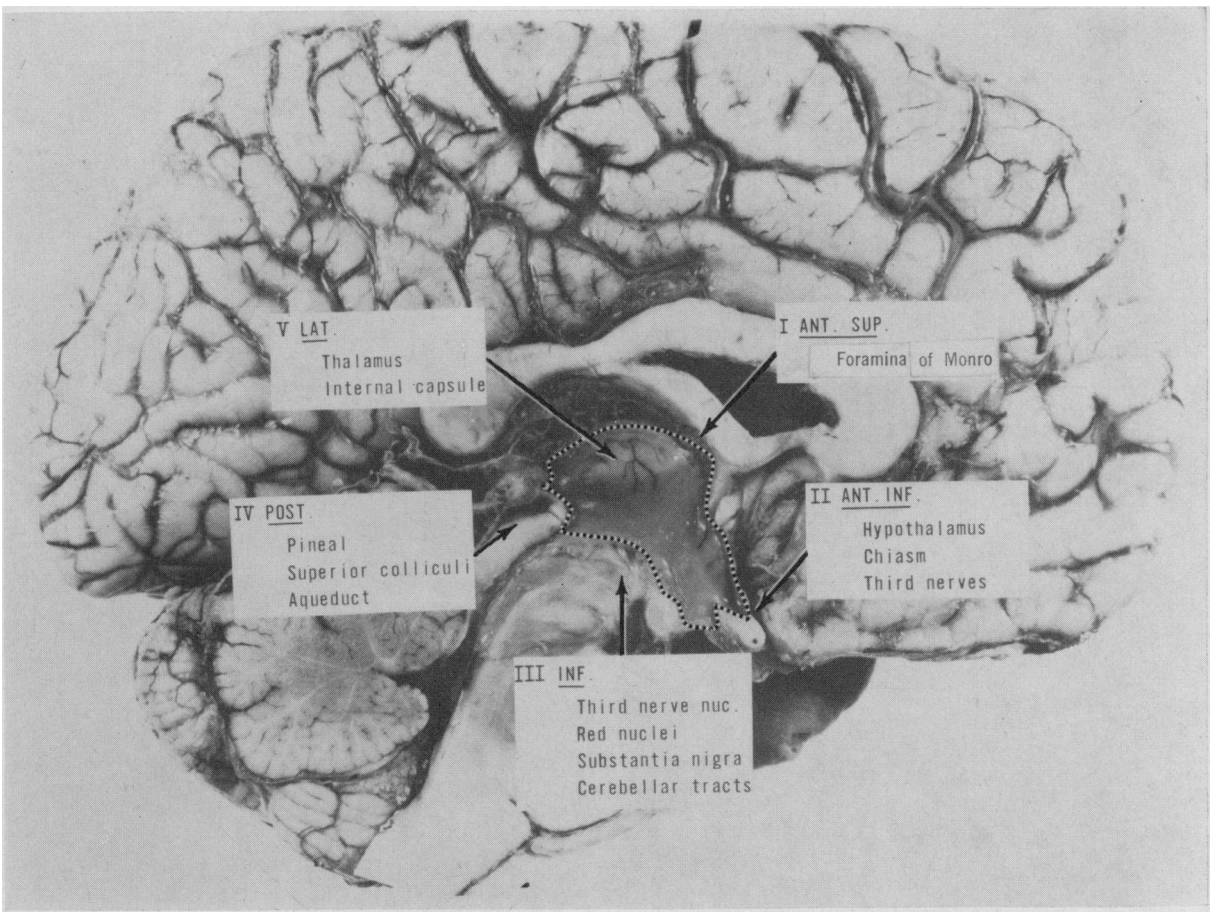


Fig. 7.—Limits of the third ventricle with enumeration of the principal symptomatic structures classified in five anatomical regions.

nerve nuclei, the red nuclei, the substantia nigra, the centre for conjugate vertical gaze. Also to be found are the posterior longitudinal bundle and the cerebellar connection fibres.

Region 4: Posterior structures.—The posterior limit of the ventricle is the pineal gland. Underneath are the superior colliculi with their pupillary and possibly optokinetic fibres. The aqueduct of Sylvius lies under the colliculi.

Region 5: Lateral structures.—Laterally the ventricle is limited by the thalamus itself in intimate relationship with the internal capsule.

The major symptoms or groups of symptoms for which tumours of the third ventricle can be responsible are summarized in Table III.[18-26]

TABLE III.—SUMMARY OF NEUROLOGICAL AND OPHTHALMOLOGICAL SYMPTOMS CAUSED BY TUMOURS OF THE THIRD VENTRICLE

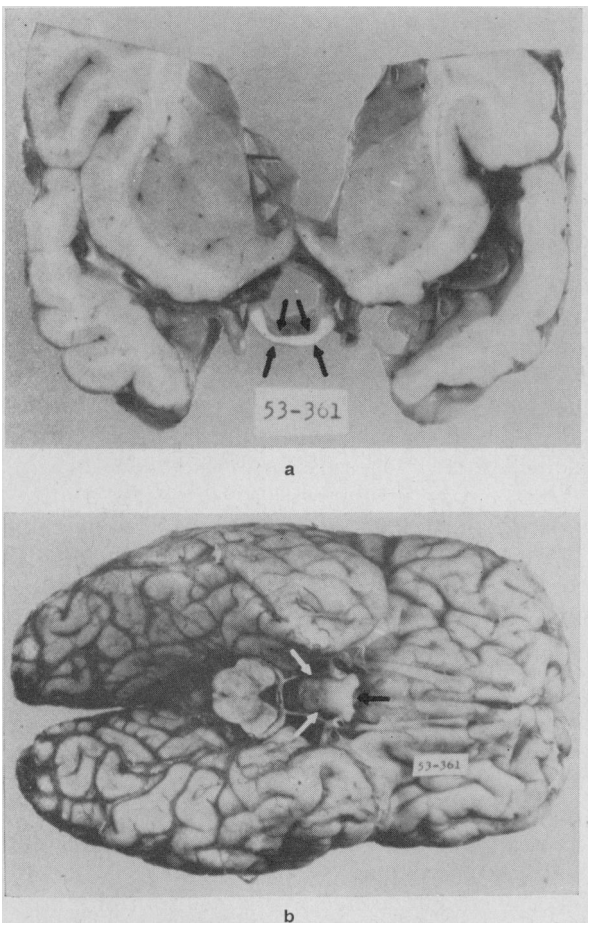
	*Symptoms**	
	Neurological	*Neurophthalmological*
Region 1		
Anterosuperior	Increased intracranial pressure Mental disturbances	Sudden loss of vision Papilledema Secondary optic atrophy
Region 2		
Anteroinferior	Endocrine Autonomic	Chiasmal interference Field defects Nerve palsies
Region 3		
Inferior	Cerebellar Extrapyramidal	Ocular palsies
Region 4		
Posterior	Pineal syndrome Increased intracranial pressure	Pupillary abnormalities Superior gaze palsy Nystagmus retractorius
	Plus added symptomatology of Region 1	
Region 5		
Lateral	Thalamic syndrome Falling spells Limb palsies Sensory disturbances	

*Grouped according to the five symptomatic regions described in the text.

II. THE PATHOPHYSIOLOGICAL BASIS FOR CLINICAL SYMPTOMATOLOGY

Increased Intracranial Pressure

(a) *Dynamics of intraventricular hypertension.*—A tumour in the ventricle may be so situated as to obstruct the drainage system, but may do so only in certain circumstances, for example, when its weight presses against the foramina. Such a mechanical effect may be brought about by certain positions of the head or body, such as stooping, bending or lying down. This is the so-called ball-valve effect of tumours of the third ventricle. Similarly, a tumour may cause



Figs. 8a and b.—Example of possible dilatation of the third ventricle under increased pressure seen in transverse section and inferior view of the brain.

chronic or periodic symptoms of blockage during its growth. Furthermore, it may continue to grow for a long time and still leave sufficient drainage space or remain in a remission phase which can last for years; a remission lasting nine years has been reported.[27]

The site of obstruction is also important. If it is at the level of the foramina of Monro (region 1), the symptomatology will be that of dilatation of the lateral ventricles. But if it is situated at the level of the aqueduct of Sylvius (region 4), the third ventricle also dilates and may press on structures surrounding it (region 2). The anterior wall, the lamina terminalis, can bulge forward, causing distortion of the chiasma. By this means a lesion situated posteriorly can cause symptoms related to anterior structures (Figs. 8a and b).

A tumour of the same size and identically located may be entirely asymptomatic in one patient, and in another may suddenly become symptomatic by producing obstruction. These patients are prone to sudden death; we have

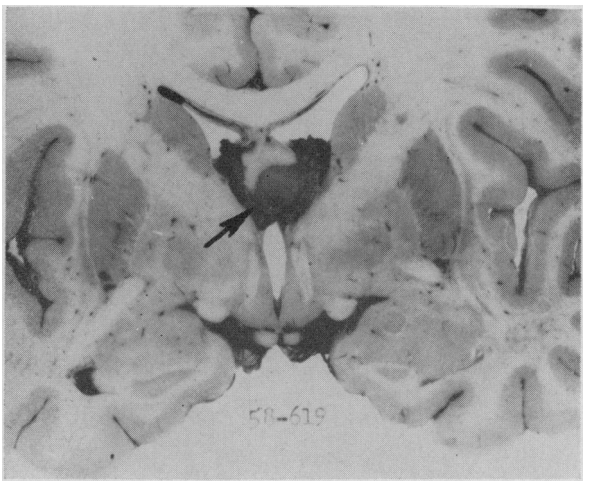


Fig. 9.—Case 1. Incidental finding at autopsy: colloid cyst.

seen this termination in our own cases, and many have been recorded in the literature. There can be a major reaction to minor trauma or technical diagnostic procedures. Sometimes acute attacks are precipitated by benign infectious disease, menstruation or other causes of systemic congestion.

CASE 1 (Fig. 9).—While undergoing abdominal surgery under general anesthesia, a 50-year-old coloured man had a cardiac arrest from which he did not recover. At autopsy a colloid cyst was found at the level of the foramina of Monro. There were absolutely no symptoms in the history referable to the presence of the cyst, which was not causing obstruction despite its size and location.

CASE 2 (Fig. 10).—A 32-year-old woman was admitted to hospital because of a sudden onset of headache and vomiting. There were no significant

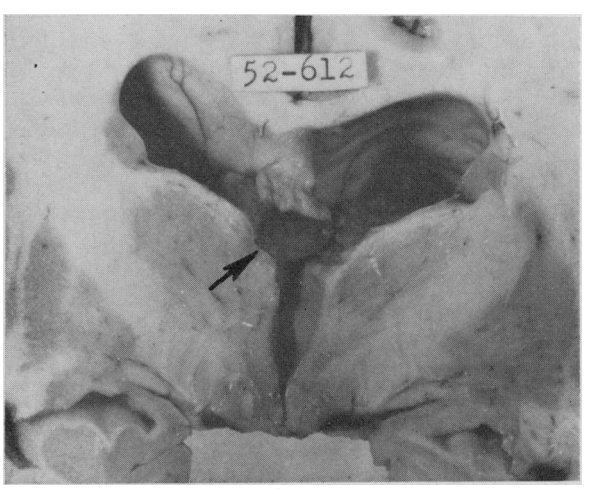


Fig. 10.—Case 2. A colloid cyst of the same size as in the preceding case; it suddenly became symptomatic and caused death.

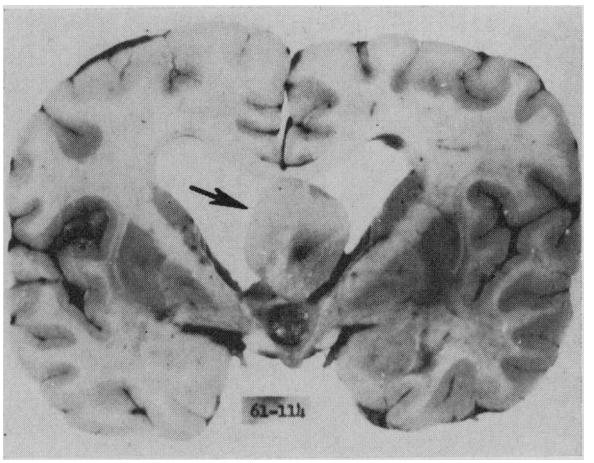


Fig. 11.—Case 3. Colloid cyst causing sudden death during pneumoencephalography.

past illnesses or symptoms. Hemorrhages were present in the right ocular fundus. She died the next day; there was no time to attempt to make a diagnosis. Autopsy revealed a colloid cyst in the area of the foramina of Monro, of about the same size as in the preceding case, but in this patient it caused obstruction. Cerebral edema and hydrocephalus were present.

CASE 3 (Fig. 11).—A 50-year-old man was admitted to hospital because of severe headaches. A pneumoencephalogram was performed, but it was unsatisfactory, technically. The following day the patient developed papilledema and some ataxia. He died while the pneumoencephalogram was being repeated. Autopsy revealed a large colloid cyst filling the third ventricle and extending towards the corpus callosum.

(b) *Signs and symptoms.*—These are always the same, whatever the cause of increased intracranial pressure, and they have no localizing value. Intense headache with or without vomiting is the most frequent presenting symptom in these tumours. One patient reported by Ford[39] had an average of one attack of headache and vomiting each month for a period of four years before seeking medical advice. The duration of the increased pressure will determine the presence or absence of papilledema and eventually, secondary optic atrophy. Transient loss or blurring of vision may be noted. Occasionally, loss of consciousness or repetitive fainting spells may be the presenting symptoms.

CASE 4.—A 24-year-old woman was admitted to hospital because of severe headaches of one month's duration. They were knife-like, occurred every day and were worse at night. Some relief was obtained by pressing both temples with her hands. She had had a few syncopal attacks. Her memory had become poor. She felt that her whole body was "like dead". Skull radiographs were normal. There was a family history of seizures, and although "a brain lesion could not be ruled out", she was thought to have a functional disorder and was dismissed. She was found dead a few days later; autopsy revealed a colloid cyst in the third ventricle.

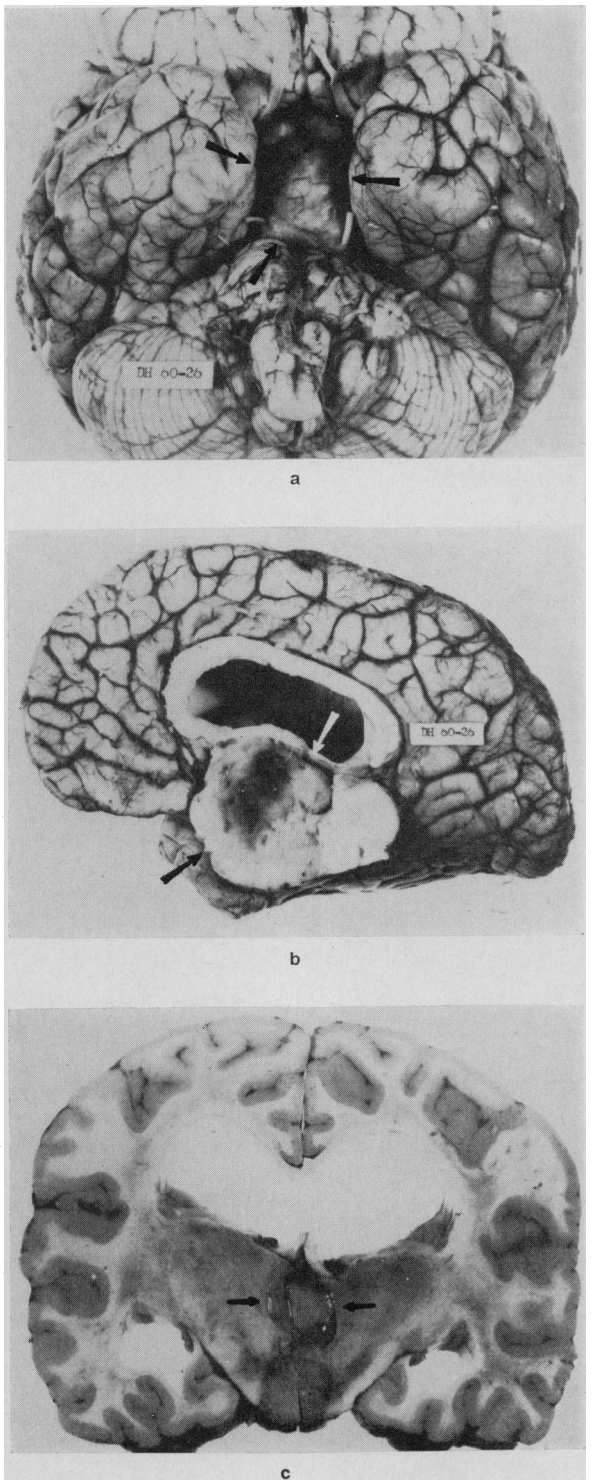
Visual Field Defects

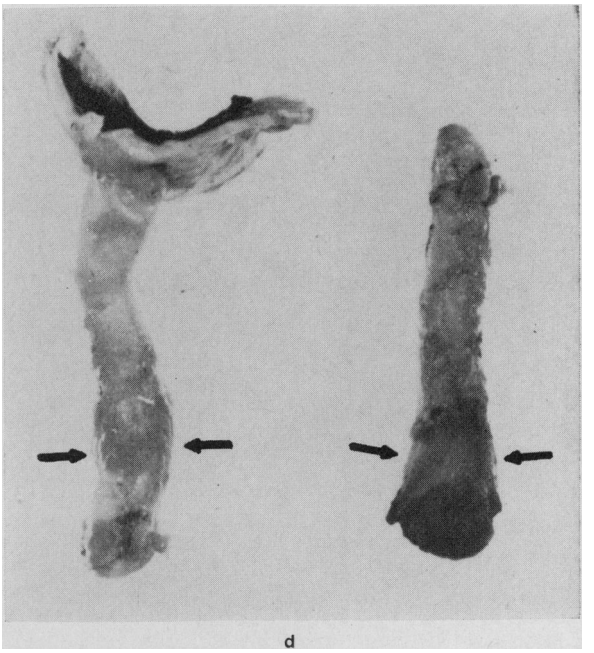
From the intimate relationship of the third ventricle to the chiasma and optic tracts, one might expect frequent modifications in the visual fields. We are amazed by the scarcity of such findings as reported by Hughes.[28] He describes a more or less asymmetrical bitemporal field defect suggesting posterosuperior chiasma involvement beginning with a peripheral contraction or a paracentral scotoma.[29] Homonymous hemianopsias,[30] bilateral pericentral scotomas and even binasal hemianopsias have been reported.[31, 32] One case reported by Kelly[18] had loss of the visual field in one eye.

Other Ophthalmological Signs

Besides papilledema and field defects, many other ophthalmological signs may be encountered. Loss of visual acuity is generally noted, as is a transient blurring of vision, although the latter can sometimes be evaluated. Optic atrophy of the chronic congestive type, resulting from long-standing papilledema, may be unilateral or bilateral. In other instances, it may be of the primary type. Motor paralysis involving all or any of the third, fourth and sixth nerves may be found. Bilateral ptosis of the upper lids with diplopia was among the presenting symptoms in a case reported recently.[32] Vertical conjugate gaze palsies are sometimes found, suggesting midbrain involvement. Similarly, *nystagmus retractorius* has been described.[34] Compression of the superior colliculi or their connections is responsible for pupillary abnormalities, which may include the Argyll Robertson type. Weisenburg[23] reports four cases of exophthalmos presumably due to compression of venous structures.

CASE 5 (Figs. 12a, b, c and d).—A 7-year-old boy was admitted to hospital because of vomiting, severe headaches and prostration of one week's duration. He had had two operations for strabismus two years previously. No ophthalmic studies were done at that time. The tentative diagnosis was "gastritis". He suddenly became drowsy and died two days later. Autopsy revealed a huge spongioblastoma involving the area of the tuber cinereum, the left thalamus and the midbrain; pus filled the entire ventricle, resulting in hydrocephalus. There was marked enlargement of the optic nerves.





Figs. 12a, b, c and d.—Case 5. Huge spongioblastoma invading third ventricle, brain stem and optic nerves, undiagnosed until autopsy.

Mental Disturbances

Progressive dementia or an organic psychic syndrome is attributed to chronic hydrocephalus.[35, 36] The patient gradually undergoes a progressive deterioration in his intellectual and socioeconomic capacities. Symptoms may be mild, such as a lack of drive or irritability. Loss of memory seems a rather common symptom.[37] Pathological sleep may also be noticed.[38]

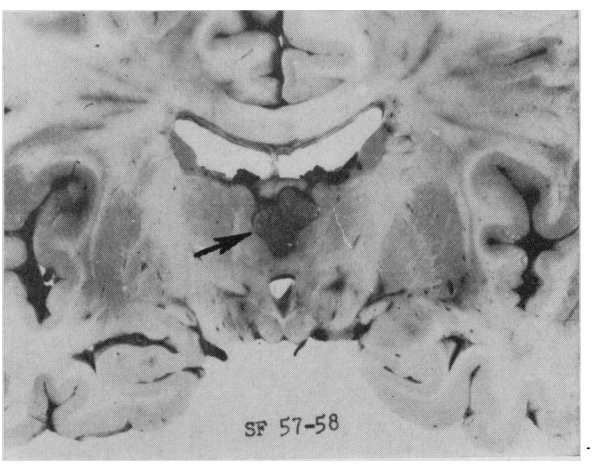
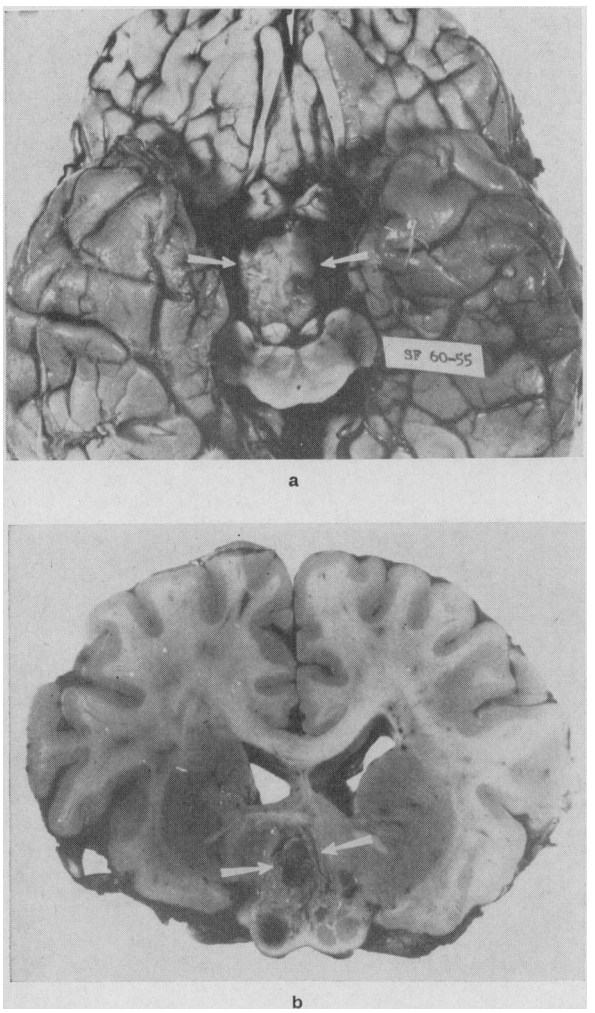


Fig. 13.—Case 6. Meningioma, diagnosed as schizophrenia.

CASE 6 (Fig. 13).—A 34-year-old man was arrested for strangeness of conduct and admitted to a mental hospital. During the previous year he had been acting queerly and had been confused and incoherent. A right exotropia was noted on admission. The diagnosis entertained was schizophrenic reaction of the hebephrenic type. The patient died 22 years later. In the third ventricle a greyish, firm tumour was found, which proved to be a meningioma.



Figs. 14a and b.—Case 7. An epidermoid tumour in a male aged 53, causing mainly psychiatric symptoms.

CASE 7 (Figs. 14a and b).—A 53-year-old man was admitted to a mental institution because of rapidly progressive mental deterioration of one year's duration. He was irritable, combative and uncooperative. He had loss of memory and was markedly confused when awake; he slept most of the time. The diagnosis was unspecified psychotic reaction. Autopsy revealed a mass filling the third ventricle and invading the neighbouring structures, including the optic tracts and chiasma. Histologically, it was an epidermoid tumour.

Falling Spells

This is a rather peculiar and relatively frequent symptom associated with these tumours. In the classical attack, the patient is suddenly stricken during normal activity. He feels his legs give way under him and he falls without any loss of his senses and with complete awareness of what is happening. He can get up and can almost immediately resume what he was doing. Many explanations are proposed for these falling spells. They may be part of a general but incomplete loss of consciousness, where the capillary circulation impairment can be just sufficient to hinder motoricity and not consciousness. They can also be the result of localized compressions or tractions on structures which by their anatomical situation are more exposed than others to the effect of the dilatation of lateral ventricles.

CASE 8 (Fig. 15).—This patient became disoriented and senile, had loss of memory and was unable to take care of himself. He had many falling spells. He died in a mental institution. An astrocytoma had grown from the septum pellucidum down into the third ventricle. There was softening of the thalamus.

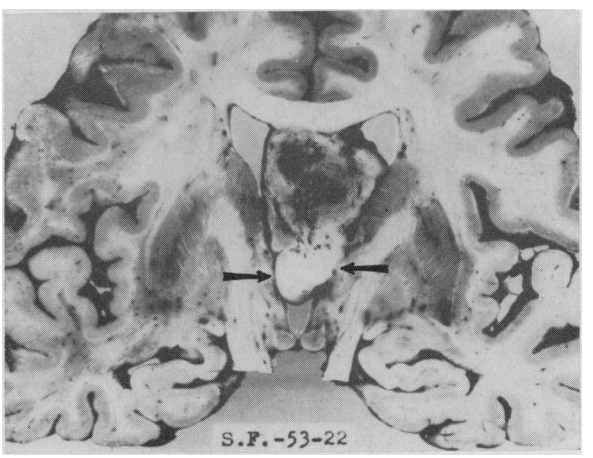


Fig. 15.—Case 8. Astrocytoma causing falling spells.

Endocrine and Autonomic Changes

Symptoms in this field vary with the localization of the tumour and the age of the patient. Precocious puberty and diabetes insipidus seem to be the most frequent.[39, 40] Hypoglycemic crises and sensitivity to insulin have been reported, as well as hyperglycemic states. Ford[39] reports a case of invasion of the floor of the third ventricle first causing somnolence, polyuria, polydipsia and fluctuations in body temperature.

Summaries of two interesting cases of tumour of the third ventricle are reported by Kahana *et al.*[33] A 40-year-old man was seen because of difficulty with his vision. A bitemporal hemianopsia was found. An arteriogram showed occlusion of the right internal carotid, and ventriculography revealed a tumour of the floor of the third ventricle. He was seen again four months later with a new series of symptoms: headache, polyuria, polydipsia, loss of memory and somnolence. Another patient, a 24-year-old

man, was seen because of polyuria and polydipsia. The examination was completely negative. Eighteen months later diplopia and paralysis of vertical conjugate gaze appeared.

An interesting case of tumour of the third ventricle is described by Penfield and Erickson[41] as "diencephalic autonomous epilepsy". The crisis may be summarized as: reddening of the face and arms, slowing of respiration, epiphora, intense perspiration, hiccough, dilatation of pupils and tachycardia. It is thought to be caused by hypothalamic compression.

Cerebellar Symptomatology

Cerebellar symptomatology in these cases may be difficult to evaluate, indicating rather posterior fossa involvement in a given case. Involvement of the superior cerebellar peduncle fibres along their pathway in the midbrain may be responsible.[42] Cerebellar gait is the most frequently reported symptom, occurring alone or associated with incoordination, ataxia and a tendency to fall in one or another direction. True rotary nystagmus and Romberg's sign have also been described.

A case reported by Ford[39] was that of a previously healthy boy, 15 years old, who developed headache, questionable papilledema and a cerebrospinal fluid pressure of 210 mm. water. Ventricular dilatation was indicative of a cerebellar tumour. An exploration of the posterior fossa was carried out, with negative results, and a course of radiotherapy was given. Two years later he developed polyuria, fatigue and irritability. Four months later, difficulties in gait and speech appeared, followed by convulsions and somnolence, and finally apraxia. Autopsy revealed a pinealoma filling the ventricular system.

Other Neurological Symptoms

A few symptoms of diverse nature which have been reported are worthy of mention. Paresis of limbs or hemiparesis is sometimes present. Hemianesthesias can be associated with so-called thalamic syndromes. Extrapyramidal rigidity and abnormal movements can also be found. Convulsive seizures may be the major presenting problems.

CASE 9 (Fig. 16).—A 51-year-old man had progressively lost all interest in his work over a period of 10 years and suffered grand mal attacks. He would awake at night with severe headache and vomiting. When admitted to a mental hospital, his right leg felt "like dead". His optic discs were pale and the right pupil was dilated. A provisional diagnosis of epilepsy due to cerebral atrophy was considered. During the next year tremor of the right side of the body and some paresis appeared. He died three years later, with the diagnosis of schizophrenic reaction of the paranoid type with somatic delusions. Autopsy revealed a large colloid cyst causing hydrocephalus, herniation of the left hippocampal gyrus, atrophy of the fornices and mamillary bodies and, finally, compression of the thalamus and hypothalamus.

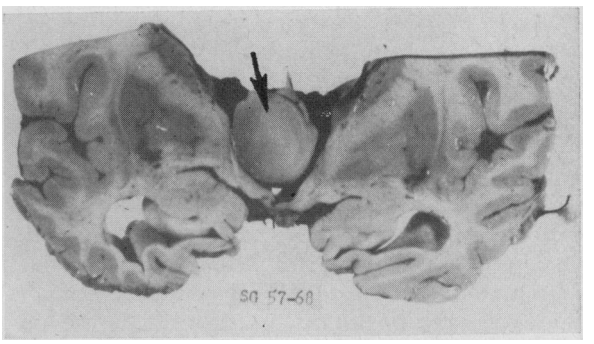


Fig. 16.—Case 9. Large colloid cyst causing chronic hydrocephalus, mental disorder and optic atrophy.

CONCLUSIONS

After reviewing the possible symptomatology of tumours of the third ventricle we can say that a "symptom complex" or a "classical history" for these tumours does not exist. Rather we find that the ventricle is surrounded by anatomical structures any one of which can produce symptoms through direct or indirect involvement. In addition, a posterior compression can provoke "anterior symptomatology" through dilatation of the third ventricle. So we are presented with a wide range of clinical possibilities. Any one symptom associated with these structures can be the presenting symptom in a given case.

It does not appear possible to diagnose the type of tumour under investigation on clinical grounds alone, although certain types of tumour can be suspected on the basis of the region presumably affected and of certain clinical manifestations.

From an entirely practical point of view, patients with a third-ventricle tumour fall into two clinical categories. The first comprises those patients whose symptoms enable a diagnosis of brain tumour to be made on the first consultation. These present no problem, as the clinical picture demands a complete and immediate work-up. The diagnosis is assured because these tumours can readily be detected by ventriculography *if it is done*. The second group of patients present entirely different and much more subtle problems because they may have fleeting symptoms or symptoms easily referable to other

etiologies. They may have complaints which may lead the ophthalmologist to think that a comprehensive radiological evaluation is unnecessary. Even simple radiography of the skull may be omitted, a serious oversight since many of these tumours show tell-tale calcifications.

The pleomorphic nature of the symptoms and their possible transience (ball-valve effect) and remission make a careful history and physical examination of supreme importance. Enough evidence may be found to ensure that the patient receives thorough technical evaluation or close observation. Sudden deaths may thus be prevented and many patients may be brought to operation at a time when surgery can confer benefit.

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